HVEM/TNFRSF14 Protein, Human (164a.a, HEK293, His)

Cat. No.:	HY-P70489
Synonyms:	Tumor Necrosis Factor Receptor Superfamily Member 14; Herpes Virus Entry Mediator A; Herpesvirus Entry Mediator A; HveA; Tumor Necrosis Factor Receptor-Like 2; TR2; CD270; TNFRSF14; HVEA; HVEM
Species:	Human
Source:	HEK293
Accession:	Q92956 (L39-V202)
Gene ID:	8764
Molecular Weight:	20-35 kDa

PROPERTIES

AA Sequence	L P S C K E D E Y P V G S E C C P K C S P G Y R V K E A C G E L T G T V C E P C P P G T Y I A H L N G L S K C L Q C Q M C D P A M G L R A S R N C S R T E N A V C G C S P G H F C I V Q D G D H C A A C R A Y A T S S P G Q R V Q K G G T E S Q D T L C Q N C P P G T F S P N G T L E E C Q H Q T K C S W L V T K A G A G T S S S H W V
Appearance	Lyophilized powder.
Formulation	Lyophilized from a 0.2 μm filtered solution of PBS, pH 7.4.
Endotoxin Level	<1 EU/μg, determined by LAL method.
Reconsititution	It is not recommended to reconstitute to a concentration less than 100 μg/mL in ddH_2O. For long term storage it is recommended to add a carrier protein (0.1% BSA, 5% HSA, 10% FBS or 5% Trehalose).
Storage & Stability	Stored at -20°C for 2 years. After reconstitution, it is stable at 4°C for 1 week or -20°C for longer (with carrier protein). It is recommended to freeze aliquots at -20°C or -80°C for extended storage.
Shipping	Room temperature in continental US;may vary elsewhere.

DESCRIPTION

Background	HVEM is widely expressed in a range of hematopoietic cells, including B cells, T cells, NK cells, monocytes and immature dendritic cells, and several non-hematopoietic cells and tissues, including the liver, kidney and lung[1]. The amino acid sequence of human HVEM protein has low homology for mouse HVEM protein. HVEM is known as the “molecular switch” models of activation and inhibition. HVEM provides an inhibitory or activating signal and bi-directionally regulates host immune function. HVEM binds to LIGHT or LIGHT-α exerts a positive stimulatory effect, stimulating lymphocyte proliferation, activation, and inducing inflammatory reactions; thus, providing a second stimulatory signal for T cell activation. Besides, the Binding of HVEM to BTLA and CD160 exerts an adverse regulatory effect,

promoting signal transduction through the ERK1/2 and PI3K (phosphatidylinositol 3-kinase)–AKT (protein kinase B (PKB)) pathways, leading to the production of IFNγ, inhibiting T- and B-lymphocyte activation and proliferation and binding of HVEM to HSV-gD, which can promote HSV infection in target cells[2][3].
HVEM is considered to be a molecular switch for immune responses, HVEM induces DCs to produce IL-10 and shows protection against experimental autoimmune myocarditis (EAM) caused by myosin[4].

REFERENCES

[1]. Ma B, et al. High expression of HVEM is associated with improved prognosis in intrahepatic cholangiocarcinoma. Oncol Lett. 2021 Jan;21(1):69.

[2]. Yu X, et al. BTLA/HVEM Signaling: Milestones in Research and Role in Chronic Hepatitis B Virus Infection. Front Immunol. 2019 Mar 29;10:617.

[3]. Rodriguez-Barbosa JI, et al. HVEM, a cosignaling molecular switch, and its interactions with BTLA, CD160 and LIGHT. Cell Mol Immunol. 2019 Jul;16(7):679-682.

[4]. Cai G, et al. Amelioration of myocarditis by HVEM-overexpressing dendritic cells through induction of IL-10-producing cells. Cardiovasc Res. 2009 Dec 1;84(3):425-33.

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